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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Sep 17	IMSworld Pharmaceutical Company Directory name change to PHARMASEARCH
NEWS 3	Oct 09	Korean abstracts now included in Derwent World Patents Index
NEWS 4	Oct 09	Number of Derwent World Patents Index updates increased
NEWS 5	Oct 15	Calculated properties now in the REGISTRY/ZREGISTRY File
NEWS 6	Oct 22	Over 1 million reactions added to CASREACT
NEWS 7	Oct 22	DGENE GETSIM has been improved
NEWS 8	Oct 29	AAASD no longer available
NEWS 9	Nov 19	New Search Capabilities USPATFULL and USPAT2
NEWS 10	Nov 19	TOXCENTER(SM) - new toxicology file now available on STN
NEWS 11	Nov 29	COPPERLIT now available on STN
NEWS 12	Nov 29	DWPI revisions to NTIS and US Provisional Numbers
NEWS 13	Nov 30	Files VETU and VETB to have open access
NEWS 14	Dec 10	WPINDEX/WPIDS/WPIX New and Revised Manual Codes for 2002
NEWS 15	Dec 10	DGENE BLAST Homology Search
NEWS 16	Dec 17	WELDASEARCH now available on STN
NEWS 17	Dec 17	STANDARDS now available on STN
NEWS 18	Dec 17	New fields for DPCI
NEWS 19	Dec 19	CAS Roles modified
NEWS 20	Dec 19	1907-1946 data and page images added to CA and CAplus
NEWS 21	Jan 25	BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 22	Jan 25	Searching with the P indicator for Preparations
NEWS 23	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 24	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency

NEWS EXPRESS	February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

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FILE 'HOME' ENTERED AT 11:03:05 ON 15 FEB 2002

=> file medline, uspatful, dgene, embase, biosis

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FULL ESTIMATED COST                                         0.15       0.15

FILE 'MEDLINE' ENTERED AT 11:03:21 ON 15 FEB 2002

FILE 'USPATFULL' ENTERED AT 11:03:21 ON 15 FEB 2002
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FILE 'BIOSIS' ENTERED AT 11:03:21 ON 15 FEB 2002
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=> s coronary artery disease

L1      109645 CORONARY ARTERY DISEASE

=> s l1 and treatment

L2       20920 L1 AND TREATMENT

=> s fibroblast growth factor

L3       55916 FIBROBLAST GROWTH FACTOR

=> s l3 and l2

L4         234 L3 AND L2

=> s (fibroblast growth factor-2)

L5        4357 (FIBROBLAST GROWTH FACTOR-2)

=> s l5 and l2

L6           7 L5 AND L2

=> d l6 ti abs ibib tot


L6   ANSWER 1 OF 7  USPATFULL
TI     Artery - and vein-specific proteins and uses therefor
AB     Arterial and venous endothelial cells are molecularly distinct from the
       earliest stages of angiogenesis. This distinction is revealed by
       expression on arterial cells of a transmembrane ligand, called EphrinB2
       whose receptor EphB4 is expressed on venous cells. Targeted disruption
       of the EphrinB2 gene prevents the remodeling of veins from a capillary
       plexus into properly branched structures. Moreover, it also disrupts
the
       remodeling of arteries, suggesting that reciprocal interactions between
       pre-specified arterial and venous endothelial cells are necessary for
       angiogenesis.
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2001:165440 USPATFULL
TITLE:	Artery - and vein-specific proteins and uses therefor
INVENTOR(S):	Wang, Hai U., Pasadena, CA, United States Chen, Zhoufeng, Pasadena, CA, United States Anderson, David J., Altadena, CA, United States

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001024650	A1	20010927
APPLICATION INFO.:	US 2001-823009	A1	20010330 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1998-85820, filed on 28 May 1998, PENDING Continuation-in-part of Ser. No. US 1998-83546, filed on 22 May 1998, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-81757	19980413 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	David E. Brook, Esq., HAMILTON, BROOK, SMITH & REYNOLDS, P.C., Two Militia Drive, Lexington, MA, 02421-4799
NUMBER OF CLAIMS:	108
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	1 Drawing Page(s)
LINE COUNT:	1613

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 2 OF 7 USPATFULL

TI Platelet derived growth factor (PDGF) nucleic acid ligand complexes

AB This invention discloses a method for preparing a complex comprised of a PDGF Nucleic Acid Ligand and a Non-Immunogenic, High Molecular Weight Compound or Lipophilic Compound by identifying a PDGF Nucleic Acid Ligand by SELEX methodology and associating the PDGF Nucleic Acid Ligand with a Non-Immunogenic, High Molecular Weight Compound or Lipophilic Compound. The invention further discloses Complexes comprising one or more PDGF Nucleic Acid Ligands in association with a Non-Immunogenic, High Molecular Weight Compound or Lipophilic Compound. The invention further includes a Lipid construct comprising a PDGF Nucleic Acid Ligand or Complex and methods for making the same.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2001:67813 USPATFULL
TITLE:	Platelet derived growth factor (PDGF) nucleic acid ligand complexes
INVENTOR(S):	Janjic, Nebojsa, Boulder, CO, United States Gold, Larry, Boulder, CO, United States
PATENT ASSIGNEE(S):	NeXstar Pharmaceuticlas, Inc., Boulder, CO, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6229002	B1	20010508
APPLICATION INFO.:	US 1997-991743		19971216 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-618693, filed on 20 Mar 1996, now patented, Pat. No. US 5723594 Continuation-in-part of Ser. No. US 1995-479783, filed on 7 Jun 1995, now patented, Pat. No. US 5668264 Continuation-in-part of Ser. No. US 1995-479725, filed on 7 Jun 1995, now patented, Pat. No. US 5674685

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Zitomer, Stephanie
LEGAL REPRESENTATIVE: Swanson & Bratschun LLC
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 30 Drawing Figure(s); 26 Drawing Page(s)
LINE COUNT: 3002
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 3 OF 7 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
TI Vascular growth factor and gene therapy to induce new vessels in the ischemic myocardium.
AB In summary, angiogenic vascular endothelial growth factor therapy either with the protein itself or with the gene encoding for the vascular growth factor is a new promising potential therapeutic alternative to patients with severe **coronary artery disease**, which cannot be treated with conventional revascularization with PTCA or CABG. The **treatment** regimes evaluated seem to be safe. However, we have to await the results of ongoing and future larger scale double-blind placebo-controlled studies of genes encoding for the vascular growth factors or of the protein formulations before we can define the role of vascular growth factor **treatment** in ischemic heart disease.
ACCESSION NUMBER: 2002042919 EMBASE
TITLE: Vascular growth factor and gene therapy to induce new vessels in the ischemic myocardium.
AUTHOR: Kastrup J.; Jorgensen E.; Drvota V.
CORPORATE SOURCE: Dr. J. Kastrup, Medical Department B, The Heart Centre, University Hospital, DK-2100 Copenhagen O, Denmark. jkastrup@rh.dk
SOURCE: Scandinavian Cardiovascular Journal, (2001) 35/5 (291-296).
Refs: 27
ISSN: 1401-7431 CODEN: SCJOFY
COUNTRY: Norway
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
022 Human Genetics
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 4 OF 7 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
TI Therapeutic angiogenesis for **coronary artery disease**.
AB A large body of evidence in animal models of ischemia shows that administration of angiogenic growth factors, either as recombinant protein or by gene transfer, can augment nutrient perfusion through neovascularization. Many cytokines have angiogenic activity; those that have been best studied in animal models and clinical trials are vascular endothelial growth factor (VEGF) and fibroblast growth factor (FGF). Clinical trials of therapeutic angiogenesis in patients with end-stage **coronary artery disease** have shown increases in exercise time and reductions in anginal symptoms and have provided objective evidence of improved perfusion and left ventricular function. Larger-scale placebo-controlled trials have been limited to intracoronary and intravenous administration of recombinant protein and have not yet shown significant improvement in exercise time or angina compared with placebo. Larger-scale placebo-controlled studies of gene transfer are in progress. Clinical studies are required to determine the optimal dose, formulation, route of administration, and combinations of growth factors and the utility of adjunctive endothelial progenitor-cell or stem-cell

supplementation, to provide safe and effective therapeutic myocardial angiogenesis. Determination of which growth factor or cells are required to optimize therapeutic neovascularization in an individual patient should be a goal of future research.

ACCESSION NUMBER: 2002017430 EMBASE
TITLE: Therapeutic angiogenesis for **coronary artery disease**.
AUTHOR: Freedman S.B.; Isner J.M.; Neely M.
CORPORATE SOURCE: M. Neely, St. Elizabeth's Medical Center, 736 Cambridge Street, Boston, MA 02135, United States. mneely222@aol.com
SOURCE: Annals of Internal Medicine, (1 Jan 2002) 136/1 (54-71).
Refs: 165
ISSN: 0003-4819 CODEN: AIMEAS
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 004 Microbiology
006 Internal Medicine
018 Cardiovascular Diseases and Cardiovascular Surgery
023 Nuclear Medicine
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 5 OF 7 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
TI Surgical and percutaneous myocardial angiogenesis induction. Part II - Neoangiogenesis.
AB Coronary artery bypass surgery and angioplasty provide symptomatic relief in patients with ischemic heart disease, but despite advancement in technique and devices, these methods are not applicable to a subset of patients with angina refractory to medical **treatment**. Bypass surgery might not be feasible because of lack of suitable conduits, diffuse coronary disease or poor distal run-off, and coronary angioplasty is sometimes not applicable due to chronic total occlusion, diffuse disease or extreme tortuosity. We have previously reviewed the available experience with laser-induced direct myocardial revascularization, one of the new potential **treatment** modalities for this patient subset. One of the potential mechanisms of action for laser **treatment** is the induction of neoangiogenesis. In the second part of our article we review the available experience with the induction of myocardial angiogenesis using different growth factors or the genes encoding for them.

ACCESSION NUMBER: 2001080439 EMBASE
TITLE: Surgical and percutaneous myocardial angiogenesis induction. Part II - Neoangiogenesis.
AUTHOR: Gimelli G.; Di Mario C.; Liistro F.; Dharmadhikari A.V.; Montorfano M.; Anzuini A.; Vaghetti M.; Puchala-Borowik M.;
Airoldi F.; Carlino M.; Tzifos V.; Maisano F.; Alfieri O.; Colombo A.
CORPORATE SOURCE: Dr. C. Di Mario, Cardiologia Interventistica, Ospedale San Raffaele, Via Olgettina 60, 20132 Milano, Italy. segreteria.emodinamica@hsr.it
SOURCE: Italian Heart Journal, (2001) 2/1 (21-24).
Refs: 30
ISSN: 1129-471X CODEN: IHJOAM
COUNTRY: Italy
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
022 Human Genetics
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English

SUMMARY LANGUAGE: English

L6 ANSWER 6 OF 7 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
TI Angiogenesis in cardiovascular disease. Current status and therapeutic potential.
AB Therapeutic angiogenesis, in the form of growth factor protein administration or gene therapy, has emerged as a new method of **treatment** for patients with severe, inoperable **coronary artery disease**. Improved myocardial perfusion and function after the administration of angiogenic growth factors has been demonstrated in animal models of chronic myocardial ischaemia. Recently, preliminary clinical trials using growth factor proteins or genes encoding these angiogenic factors have demonstrated clinical and other objective evidence of relevant angiogenesis. Thus, therapeutic angiogenesis has the potential to extend **treatment** options to patients who are not optimal candidates for conventional methods of myocardial revascularisation.
ACCESSION NUMBER: 1999318613 EMBASE
TITLE: Angiogenesis in cardiovascular disease. Current status and therapeutic potential.
AUTHOR: Sellke F.W.; Simons M.
CORPORATE SOURCE: Dr. F.W. Sellke, Division of Cardiothoracic Surgery, Beth Israel Deaconess Medical Center, Harvard Medical School, 330 Brookline Avenue, Boston, MA 02215, United States. fsellke@bidmc.harvard.edu
SOURCE: Drugs, (1999) 58/3 (391-396).
Refs: 34
ISSN: 0012-6667 CODEN: DRUGAY
COUNTRY: New Zealand
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 7 OF 7 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
TI Fibroblast growth factor-mediated angiogenesis for the **treatment** of ischemia. Lessons learned from experimental models and early human experience.
AB Fibroblast growth factors (FGFs) are a family of nearly twenty heparin-binding growth factors. They are widely distributed throughout the body, but their activity is tightly controlled. This review will focus on fibroblast growth factor-1 /FGF-1) and **fibroblast growth factor-2** (FGF-2) which have been studied extensively in vitro and in vivo. These two growth factors stimulate the proliferation of cells of mesenchymal origin, including the three principal vascular cell types: fibroblasts, endothelial cells and smooth muscle cells. The molecular characteristics of these growth factors, their receptors, distribution, function, pharmacokinetics, hemodynamic effects and toxicity are reviewed herein. The experimental evidence for the potential for FGFs as therapeutic agents for the **treatment** of progressive myocardial ischemia, acute myocardial ischemia, and peripheral limb ischemia is also analyzed. It is not known to what extent the results of animal studies can be extrapolated to humans with ischemic cardiovascular disease. Clinical trials have been initiated, and there is a growing hope that the pharmacologic use of growth factors will represent a viable therapeutic alternative for patients with ischemic cardiovascular disease.
ACCESSION NUMBER: 1998372756 EMBASE

TITLE: Fibroblast growth factor-mediated angiogenesis for the treatment of ischemia. Lessons learned from experimental models and early human experience.

AUTHOR: Goncalves L.M.
CORPORATE SOURCE: L.M. Goncalves, Cardiology Branch, National Heart, Lung/Blood Institute, National Institutes of Health, 10 Center Drive, Bethesda, MD 20892-1518, United States. goncalvl@gwgate.nhlbi.nih.gov

SOURCE: Revista Portuguesa de Cardiologia, (1998) 17/SUPPL. 2 (11-20).
Refs: 103
ISSN: 0304-4750 CODEN: RPCADZ
COUNTRY: Portugal
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English; Portuguese